Clinical Osteopathically Integrated Learning Scenarios

Patient with Chronic Obstructive Pulmonary Disease

Prepared by: AACOM's Educational Council on Osteopathic Principles



Part 4: Chapter 1

Pulmonary Clinical Osteopathically Integrated Learning Scenario:

Patient With Chronic Obstructive Pulmonary Disease



Description

his Clinical Osteopathically Integrated Learning Scenario (COILS) focuses primarily on the palpatory evaluation and supportive osteopathic manipulative treatment for a patient with chronic obstructive pulmonary disease (COPD).

The COILS is divided into two sections:

Section One

The **Roundtable Discussion Workshop** includes a discussion and evaluation of the patient's case history, diagnosis, pathophysiology, osteopathic principles involved, functional anatomy, treatment options, contraindications, and (if time permits) a demonstration of manipulative treatment techniques applicable to the patient's homeostatic needs

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Section Two

The **Patient-Based Application Workshop** is the supervised application of manipulative treatment techniques for a patient with this diagnosis. The workshop is designed to evaluate the student's or physician's diagnostic and psychomotor skills when providing osteopathic manipulative treatment for an actual (or simulated) patient.

If time permits, the instructor may deliver the entire two-section program at one time. Ideally, however, Section One should be taught several days before Section Two to allow time for the student or physician to review and practice appropriate techniques. If an actual patient is not available for Section Two, a simulated patient may be used.

Section One: Roundtable Discussion Workshop I. Description

This section is a roundtable-type presentation with discussion on the osteopathic approach to the treatment of a patient with COPD.

II. Cognitive Components

A. Case Presentation

A 56-year-old male patient presents with complaints of a worsening shortness of breath, productive cough over the past week with yellowish–green sputum, sore muscles, and fa-tigue. He states that he may have a low-grade fever but has not had any chills. Over the past few years, the patient has had a recurrent daily cough with increased mucus production. He has a 30 pack/year smoking history but quit 6 months ago. Since then, has been on 2 liters of O2 by nasal cannula, which he requires to comfortably breathing. Howev-er, he often feels that is his breathing is inadequate and feels "hungry for more air". He is able to lie flat without increased discomfort.

The patient has a history of hypertension and recurring pneumonia. He currently take tiotropium bromide inhaler used once a day, salbutamol inhaler 1-2 puffs q4-6 hours as needed for acute exacer-bation of respiratory symptoms, spironolactone 25 mg/day, O2 by nasal cannula 2/liters/ minute and multivitamin daily. Only known allergies are seasonal and penicillin, which causes hives and further breathing difficulties. His diet is high in red meats, dairy, carbohydrates. His past surgeries include Tonsillectomy and adenoidectomy as a child.

The patient is a retired data clerk, a position in which he worked for more than 35 years. Patient has been married for 20 years and has 2 teenage children. He drinks 3-4 beers a day and denies illicit drug use. Both parent are deceased and had hypertension, diabetes and were heavy smokers. The father died in his 40's of lung cancer. The mother died 65 from hear disease complications.

Physical Examination

Vital signs:	Temperature, 100.4° F; Blood Pressure: 150/86; Pulse, 90; Respiratory Rate 24;
	Height: 5' 10"; Weight: 210 lb
General:	Mildly uncomfortable
Head:	Rounded face with large amount of fatty tissue
Eyes:	A few small retinal hemorrhages noted; "copper wiring" of the vessels
Nose:	Turbinates without any mucus
Throat:	Red oropharynx without any exudates
Neck:	No lymphadenopathy appreciated

Cardiac:Rapid and regular heartbeat, without an S3 notedLung:Dull, decreased breath sounds with scattered rales notedExtremities:Early clubbing of the fingers noted with loss of lovibonds angle but without
distal tufting; 1+ lower-extremity edema noted

Osteopathic Structural Examination

- Increased chest wall A-P diameter with barrel shape. Marked respiratory diaphragm restriction is notable beneath both costal margins. Chest wall compliance and excursion during respirations are markedly reduced bilaterally with ribs 3-7 exhibiting inhalation dysfunctions bilaterally.
- T3 extended, rotated left, sidebent left
- T7 flexed, rotated right, sidebent left.
- Increased abnormal tissue texture changes are noted from T3-T8 bilaterally with decreased sidebending.
- Exquisitely tender bilateral Chapman points are palpated anteriorly at the 3rd and 4th costal interspaces adjacent to the sternum, and posteriorly in the paraspinals between T3-T4 and T4-T5 transverse processes.
- Head and neck are forward of the gravity line with tightened and hypertrophied bilateral sternocleidomastoid muscles.
- Middle and anterior scalene musculature is hypertonic with bilateral elevated first ribs.
- OA is flexed, rotated left, sidebent right.
- AA is rotated right.
- C2 is flexed, rotated left, sidebent left.
- L1 and L2 are markedly restricted in rotation to the right and left.

Diagnostics

Labs:	Pulse oximet	try is 88% on 2L	/min by nasal canr	ula.(1, 2); alpha	a1-trypsin levels normal
CBC:	WBC	11.0	(3.5-10.5)	1000/mm3	high
	RBC	5.0	(3.90-5.03)	mega/mm	
	Hgb	15.0	(12.0-15.5)	gm/dL	
	Hct	44.0	(36.3-43.4)	%	high
	MCV	92.5	(81.6-98.3)	fL	
	MCH	30.4	(27.0-31.2)	pg	
	MCHC	32.9	(31.8-35.4)	%	
	RDW	13.7	(11.9-15.5)	%	
	Platelet	236.0	(150.0-400.0)	1000/mm3	
	MPV	8.5	(7.4-10.4)	fL	
	Neutr#	4.5	(1.4-6.5)	1000/mm3	
	Lymph#	4.0	(0.6-3.4)	1000/mm3	high
	Mono#	0.5	(0.1-0.5)	1000/mm3	

Eos#	0.1	(0.0-0.7)	1000/mm3
Baso#	0.0	(0.0-0.2)	1000/mm3

X-rays: Increased bronchovascular markings, lung hyperinflation, flattened respiratory diaphragm and cardiomegaly.

ECG: Sinus rhythm, right axis deviation, right ventricular enlargement

PFTs: Post-bronchodilator FEV1 40% of predicted; FEV1/FVC 50% of predicted

B. Pathophysiology

- 1. One of the most common causes of COPD is smoking cigarettes. The toxic chemicals in cigarette smoke cause permanent deleterious inflammatory changes to the lung resulting in a progressive irreversible obstruction of air flow. Patients exhibit a combination of varying degrees of emphysema and chronic bronchitis. Emphysema is characterized by a decrease in structural integrity of the small alveoli and elastic recoil properties of lung parenchyma. Chronic bronchitis is characterized by inflammatory cell infiltration and response resulting in small airway fibrosis, hypertrophy of the mucosa with more active goblet cells and associated mucus accumulation and plugging. Sustained sympathetic tone results in bronchial dilation, and the production of a sticky, thick mucus, which is difficult to clear. The cilia are less efficient in their ability to move this type of mucus. Thick, "stagnant" mucus traps bacteria in a protein-rich media for bacterial colonization and secondary infection of the lungs.
- 2. The decreased efficiency of the ciliated epithelial cells to clear mucus becomes a significant problem. Smaller airways become plugged with this mucus, and inflammatory mediators accumulate. If these airways are not cleared by coughing or mechanical methods, bacterial colonization increases.
- 3. Reactive bronchospasm also reduces the luminal size of the bronchi. Hypertrophy of respiratory epithelium and mucus plugging further the obstructive phenomenon. If pulmonary function declines further and respiratory effort increases further, cor pulmonale may develop.
- 4. Forced exhalation collapses small bronchi and further traps air. Forced vital capacity diminishes. The patient begins to exhale with pursed lips to maintain high intrabronchial pressure, trying to prevent bronchial collapse.
- 5. With a large amount of body energy needed to maintain breathing, any further oxygen requiring activities, such as ambulating, can have significant consequences.
- 6. The lungs typically get their sympathetic pre-ganglionic innervation from T2-T7. The viscerosomatic reflexes resulting from irritation of the lungs due to COPD are typically not localized unless the pleura are involved. Activation of general somatic afferents from the lungs results in widespread tissue texture changes which can extend from the cervical region through the lower thoracic area

C. Functional Anatomy

Includes knowledge of structure and physiology necessary to properly carry out the supportive osteopathic manipulative treatment.

- 1. There is an increase in total lung capacity as the thoracic cage expands to accommodate the trapped air caused by the expiratory dysfunction. The person has a "barrel-chested" look as a result. The thoracic cage assumes the position of maximal inhalation. It has increased AP and lateral diameters. With increased work of contraction, resting diaphragmatic tone is increased, and the diaphragm becomes flattened.
- 2. The AP curves of the spine increase, placing mechanical stress on its transition areas.
- 3. Overuse of the accessory muscles—the scalenes, abdominals, and intercostals—is common and can lead to cervical, thoracic spinal or rib dysfunction, resulting in chronic cervical or thoracic pain.
- 4. Poor diaphragmatic function may be a localized phenomenon or related to cervical somatic dysfunction. Increased air trapping expands the volume of the lung resulting in flattening of the diaphragm and less excursion during respiration. Because the motor innervation of the diaphragm is through the C3, 4, and 5 nerve roots, compromise of these nerve roots by cervical somatic dysfunction can impair impulses through the nerves and hence decrease the contraction of the diaphragm.

D. Goals for Osteopathic Manipulative Management

Includes a review of treatment pearls; a general plan for manipulative treatment of the patient; and a discussion of treatment options, contraindications, and plans for follow-up evaluation and treatment.

- 1. Treat somatic dysfunctions found from T2-T7 to decrease the negative sympathetic influence to the pulmonary tract from facilitated cord segments. With paraspinal inhibition and active rib raising, the patient will expectorate a significant quantity of sputum and thus immediately improve. Decreasing sympathetic tone also results in production of a thinner mucus from the respiratory epithelium.
- 2. Treat upper cervical somatic dysfunction and occipitomastoid sutures to normalize parasympathetic function from the vagus. Pre-ganglionic fibers from the vagus synapse with parasympathetic ganglia located in the walls of the airways. Post-ganglionic cholinergic fiber stimulation to the bronchial smooth muscle, mucous glands and blood vessels results in bronchoconstriction, increased serous mucous secretion and vasodilation, respectively.
- 3. Improve the motion of the thoracic cage and ribs. The thoracic cage is in maximal inhalation, which results in the patient having no inspiratory reserve. Assisted exhalation techniques are recommended and are the most effective.
- 4. Decrease the need for accessory muscle action and respiratory effort.
- 5. Treat infections aggressively, including appropriate atibiotics and postural drainage.

- 6. Improve lymphatic return and efficiency of diaphragmatic movement. The thoracic inlet and the thoraco-abdominal diaphragm need to be treated prior to mobilizing lymphatic flow.
- 7. Treat other somatic dysfunctions that may impair respiratory function.
- 8. Note that exercise improves the patient's sense of well-being, even though measured pulmonary function may not change. Diaphragmatic breathing exercises take a load off accessory respiratory muscles and improve the "respiratory pump."
- 9. Treat the bronchial tree and airways utilizing visceral manipulation.
- E. Contraindications and Cautions Regarding Treatment

See contraindications to treatment, Foundations, pp. 1015–1024.

- 1. Lymphatic pump techniques that are performed too vigorously can exacerbate hypoxemia, coughing, and pulmonary obstruction.
- 2. Lymphatic pump techniques can worsen respiratory obstructive conditions. The thoracic cage is too stiff for the classical thoracic lymphatic pump to work. Classic thoracic lymphatic pump for a patient with severe reduced pulmonary reserve can cause syncope. Assisted exhalation technique is recommended.
- 3. COPD patients can develop steroid-induced osteoporosis. Caution should be used with HVLA techniques or sudden compression/decompression techniques on "barrel-chested" patients.

F. Instructor's Notes

Personal clinical pearls and lessons learned from previous COILS presentations.

- 1. Robert Kappler, DO found that 100 percent of the patients involved in a study of the effects of OMM/OMT on COPD patients had dysfunction at T3 right. Treatment of the right T3 with direct action techniques was effective in improving symptoms.
- 2. Postural drainage was unnecessary when OMM/OMT was being performed as part of the patient's care.

III. Psychomotor Components

If time permits, this part can be carried out on a simulated patient.

1. Practice palpatory diagnosis. (See techniques under Section D above.) Diagnostic procedures should include attention to the following regions: cervical spine, upper thoracic spine and ribs, thoracic inlet, anterior chest wall, thoraco-abdominal diaphragm, thoracic cage compliance, tissue texture palpation and evaluation, psoas hypertonicity, and Zink whole-body fascial pattern.

- 2. Demonstrate key treatment techniques in the involved body regions, including seated articulatory techniques, inhibitory pressure, release techniques for the upper thoracics and ribs, OA myofascial release or indirect techniques, cervical techniques, and treatment techniques with the patient in the semi-Fowler's position.
- 3. Evaluation of a plan for treating the patient in the appropriate position, localization of gentle forces, and activation.

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V. Examination Questions

These multiple-choice questions involve the treatment of a patient with COPD.(denotes correct answer)*

- 1. A 64-year-old patient with chronic bronchitis has a productive cough with thick mucus. How will treating the upper thoracic spine with OMT benefit this patient?
 - A. It will stimulate the vagus nerve and cause bronchodilation.
 - B. It will inhibit the cough reflex, providing symptom relief.
 - C. It will thin the mucus by decreasing sympathetic tone.
 - D. It will mechanically cause a flattening of the diaphragm, which increases negative intrathoracic pressure.
 - E. It will directly stimulate the phrenic nerve and cause better diaphragm motion.
- 2. A medical student asks you why a patient with COPD has a "barrel chest." You respond,
 - A. Air trapping from COPD causes the thoracic cage to expand due to an increased total lung capacity.
 - B. COPD patients retain air, causing exhaled rib lesions, which produce the characteristic increase in AP diameter.
 - C. The muscles of the thoracic cage become ischemic and stiff.
 - D. The scalenes do all the work of breathing and pull the upper ribs superiorly.
 - E. Chronic viscero-somatic input to the body wall causes fibrosis of the tissues.

- 3. After evaluating a patient's respiratory biomechanical problems at the bedside, you find the most significant findings were a poorly functional diaphragm, a stiff thoracic cage (barrel chest), pursed lip breathing, and scalene hypertonicity. You decide to review the chest x-ray and find a small heart, hyperinflation, and a flat diaphragm. Your provisional diagnosis is
 - A. Emphysema
 - B. Asthma
 - C. Chronic bronchitis
 - D. Pulmonary embolic disease
 - E. Congestive heart failure

4. In a patient with chronic bronchitis, which manipulative procedure is most appropriate to induce a cough producing copious amounts of sputum?

- A. Seated direct HVLA to the upper thoracic area
- B. Supine counterstrain to lumbar spine
- C. HVLA to mid-cervical spine
- D. Prone soft tissue to thoracolumbar area
- E. Lumbar soft tissue or articulation with the patient on their side

5. The pathology of the bronchial mucosa associated with chronic bronchitis reveals?

- A. Increased mucus-secreting goblet cells
- B. Squamous metaplasia
- C. Decrease in the number of functional cilia
- D. Thinning of mucosa
- E. Areas of bronchi denuded of mucosa

6. Forced vital capacity is less than slow vital capacity in a COPD patient because:

- A. Small bronchi collapse with a large expiratory effort, trapping air.
- B. Forced vital capacity expels too much CO2, causing respiratory acidosis.
- C. The patient is too fatigued to provide an adequate expiratory effort.
- D. Forced exhalation produces acute hypotension.
- E. Rib cage restriction prevents forced exhalation.

Section 2: Patient-Based Application Workshop

I. Description

This section includes the practical application of osteopathic treatment techniques to support the patient with COPD.

II. Psychomotor Components

(Refer to Section 1 for regions of the body that are involved.)

- 1. Examination of the patient using TART, including postural screen, palpation, segmental motion testing, and diagnosis of somatic dysfunction.
- 2. Application of philosophy and treatment technique.
- 3. Re-evaluation of the patient after treatment is completed to assess results. If a simulated patient is used, then the student or physician should verbalize length of treatment and future treatment goals.

III. Cognitive Components

- 1. Documentation in the medical record.
- 2. Post-treatment discussion.

Note. It is recommended to use the standardized outpatient form included in each of these chapters for documentation.

Physician:			Date:
Title: [] Resident (Specialty)			
[] Intern [] OMS III	[]0[VIS IV	
	nt with C	hronic O	st of Osteopathic Principals bstructive Pulmonary Disease
CRITICAL ACTION	COMF Yes	PLETED No	COMMENTS
Become familiar with the patient's history physical examination findings, laboratory and other diagnostic findings.	163		
Perform an osteopathic structural examination.			
Determine significant areas of somatic dysfunction.			
Determine body region(s) to be treated with OMT.			
Apply OMT to at least the body region determined to be the most in need of treatment at present time.			
Treat other significant somatic dysfunctions if feasible.			
Document treatment and immediately observable effects.			

Trainer:___

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HIEF COMPLAINT:						
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